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ABSTRACT

This monograph presents a general introduction to the history, classification, and characteristics of mental retardation. It begins with a discussion of the history of mental retardation from ancient Greece and Rome to the present. The beginnings of special education are traced to the early 19th century in Europe. Major influences in treatment of the mentally retarded in the United States during the 19th and early 20th centuries are summarized. Changes in definitions of mental retardation are traced from 1845 through 1983. A section on classification addresses degrees of mental retardation and provides an extensive discussion of various etiologies including infection or intoxication, trauma or physical agent, metabolism or nutrition, gross brain disease, unknown prenatal influence, chromosomal abnormality, gestational disorders, retardation following psychiatric disorder, and environmental influences. Characteristics of individuals with mental retardation are specified for mild mental retardation, moderate mental retardation, severe mental retardation, and profound mental retardation. Appropriate instructional goals are also offered for each level. (Contains 93 references.) (DB)

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MENTAL RETARDATION IN PERSPECTIVE

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1993

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HISTORY OF MENTAL RETARDATION

Sociopolitical factors have influenced the treatment of those considered unworthy or different since the earliest times. In many societies, even the accusation or potential of being different was sufficient grounds for maltreatment of the individual. Early in the history of mental retardation, the rights of the group often took precedence over the rights of the individual. When carried to the extreme, the right of a handicapped baby to live was frequently denied by the group. Infanticide was practiced by societies either struggling to survive or characterized by a harsh nature. Victims included those who were deformed, diseased, female, or bastards. In the case of mental retardation, it is difficult to speak authoritatively because the condition was not defined as a discrete entity until the 19th century. Prior to the nineteenth century, it was typical to group individuals who were considered to be deformed, degenerate, mentally ill, physically handicapped, or mentally retarded into a single category. It is probable that only the small percentage of today's mentally retarded population labeled severely and profoundly retarded were considered historically as unworthy. This is because those who are retarded to a greater degree are more likely to exhibit physical involvement to a greater extent.

Approaches to working with the mentally retarded were often based on ignorance, fear, and superstition, leading to extreme treatment. Deformed infants who were left to the elements or thrown off cliffs were, in some cases, mentally retarded. Less afflicted individuals who were not exterminated were sometimes treated by exorcism, massages, baths, drugs, bloodletting, and trepanning. Trepanning appeared to be an early form of neurosurgery designed to affect an individual's behavior. The procedure involved cutting or boring a hole in an individual's skull. Trepanning was occassionally performed more than once on the same individual. This generally crude procedure often killed the victim or left the skull in a noticeably deformed state. It is likely that the procedure did affect the individual's behavior and was, therefore, a practice that continued for some time.

As societies became more established, there was less need to exterminate those who were not fully acceptable or productive. A number of differentiated roles became available to the handicapped as the social structure changed. This does not mean, however, that unfortunate people were fully integrated. Plato (427-347 BC) suggested in The Republic that goodness, truth, and beauty are related to each other. Deviations from the norm were errors related to evil and ugliness. He also suggested that children of inferior parents and those that are born defective be hidden away. On the other hand, Confucius (551-479 BC) urged people to be

kind and help those of weak mind. Harms (1976) suggests that the followers of the Egyptian god Osiris may have protected mentally retarded and other disabled children.

During the Roman Empire, treatment of the mentally retarded could be characterized generally as inhumane. Moderately retarded people were probably added to the list of unfortunates. By the second century A.D., defective people became popular sources of amusement in wealthy households. The statesmen Seneca (4 BC - 65 AD) wrote about Herpaste, a female fool his wife kept, who had recently lost her sight. Listening to Herpaste plead with her keeper to take her out of the house because the house was dark is an example of the kind of humor enjoyed in that era. By 200 A.D., it was possible to go to a special market in Rome which specialized in displaying people one might see in contemporary carnivals and sideshows. Mentally retarded people were also confined to hospitals designed to care for a wide range of people. Treatment in the hospitals was characterized by the belief that deprivation of light, food, and water would be therapeutic. In addition, patients could also be subjected to drugs, whipping, chaining, and in some cases, castration.

Greek and Roman literature, the Bible, the Talmud, and the Koran contain references to handicapped people. Juvenal (60-140 AD) wrote a satire in which he featured endemic goiter, a condition associated with cretinism and subsequent mental retardation. Pollo suggested that goiter was due to bad drinking water, but little else was accomplished until the 16th century. Early Christians such as St. Paul (1 Thessalonians 5:14) urged comfort for the weak of mind. In the fourth century, St. Nicholas of Myra urged love, compassion, and tender care to improve the condition of the mentally retarded and mentally ill. He demonstrated his commitment by gathering, sheltering, and teaching a group of individuals according to his precepts. Mohammed (569-622) felt that those who were weak in understanding should be maintained. This treatment was certainly not the norm, however.

During the Middle Ages (500-1450), religious houses and foundling homes were established to offer physical care to handicapped people. Treatment included the wearing of amulets, drinking of herbal concoctions, dunking in sacred pools, whipping, tying people to church pillars, and the use of fright. A notable exception was the Mansur hospital established in Cairo in the 6th century to provide humane treatment in a soothing atmosphere. The treatment that the mentally retarded received during the Middle Ages varied from treating them as innocents and letting them roam at will, to providing them with humane treatment, to exorcising the devil from them, or to locking them away. Some thought that the parents of disabled children were being punished for their sins. This lack of knowledge led people to look upon the mentally retarded with indifference, loathing, fear, and

disgust. In the 12th century in Prussia, the mentally retarded were jailed. It was 1220 before de Vitry identified cretinism as a form of mental deficiency. Three centuries were to pass before a serious attempt to describe the condition of cretinism was attempted. In the 13th century, a civil institution was developed in Geel, Belgium to render humane treatment which is still in existence. Citizens adopted the mentally retarded and integrated them as much as possible into their daily activities.

Much more typical was treatment in Hamburg and London. In 1346 in Hamburg, the mentally retarded were confined to a tower called the Idiot's Cage. Institutional treatment was developed in London at the Hospital of St. Mary of Bethlehem in 1247. The hospital was converted to an asylum in 1377 and the name gradually became contracted to Bedlam, a term still associated with the negative aspects of institutionalization. Mentally retarded patients were confined with the mentally ill, poor, criminals, and degenerates and controlled by fear. Adding to the problems of the patients was the belief that the mentally retarded and mentally ill did not suffer hunger, cold, or pain. As a consequence of this belief, high mortality rates at this and other institutions were common.

Religious beliefs have greatly influenced the treatment of the mentally retarded. During the Inquisition in the 16th century, the mentally retarded were occassionally executed. Martin Luther and John Calvin believed that the mentally retarded had lost their souls. A popular view was that the devil possessed the space which the soul ordinarily occupied. Mental retardation was one manifestation of possession. These were godless creatures who could be executed if the devil could not be driven out by physical torture. Ideas such as this were supported by the belief that everyone must take total responsibility for his actions. Those who could not were unworthy in the eyes of God.

Into the Renaissance, the mentally retarded were called Simple Simons, village idiots, fools, gulls, bumpkins, dolts, and asses. They had become the playthings of the wealthy, royalty, and the famous. Charles the 5th of France, the astronomer Tycho Brahe (1546-1601), Pope Leo, Phillip the 4th of Spain, and the Aztec Montezuma kept court jesters, scapegoats, and fools. Brahe thought that his mentally retarded dwarf uttered divine revelations.

The sixteenth century was marked by a change which included investigations of etiology as well as religious interpretations of mental retardation. About 1530, Paracelsus developed a theory of how fools were born. He thought that artisans or vulcans took the conceptus furnished by the father and mother and sculpted an infant. If the vulcan who sculpted the infant was bad, a fool or cripple was produced. Bad vulcans resulted from the fall of Adam. Besides this religious interpretation, Paracelsus described cretinism and noted the association between cretinism and

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endemic goiter in locations where the water was "peculiar". This environmental attribution was the first significant contribution since the thirteenth century to the understanding of cretinism. In 1534, Anthony Fitz-Herbert attempted to scientifically define an idiot, a term which originally came from ancient Greece. The term evolved from the description of a common man, to that of an unsophisticated layman without professional knowledge, to an ignorant individual, and finally to someone who was mentally deficient. In spite of the efforts of Fitz-Herbert, misconceptions about mentally retarded people persisted. In 1602, Platter added to the work of Paracelsus by describing cretinism as congenital feeblemindedness, but did little else beyond description of the condition.

While debate and study proceeded, the needs of the mentally retarded had to be met. St. Vincent de Paul (1581-1660) gathered together a few idiots and attempted to teach them. As was true in the past, this humane treatment was the exception to the rule. Treatment and conditions in both the religious and relatively new public institutions can, at best, be described as atrocious. St. Vincent de Paul and his Sisters of Charity established the Bicetre in Paris in the middle of the 16th century. Its original purpose was to provide shelter, food, clothing, and protection. By the end of the 18th century, the center had evolved into a collection of narrow, wet, vermin-infested cells. Attendants were brutes recruited from the prisons who controlled the residents by burdening them with chains and tying them with ropes. Treatment had deteriorated to the level associated with Bedlam in the fourteenth century.

During this time, study of the condition continued. Willis (1621-1675) attributed retardation to parents who were intemperate, too young, too old, epileptic, or too much given to study. He felt that a team of teacher and physician would be needed to help the retarded child. Willis was the first to make a clear distinction between schizophrenia and mental deficiency.

John Locke (1632-1704) in 1690 attempted to distinguish between idiocy and insanity by refining the theory of perception proposed by Descartes (1596-1650). Both Locke and Rousseau were sensationalist philosophers who believed that the mind was a blank tablet at birth and developed through sensory experiences and associations of those sensations, i.e., reflective thinking. Sensationalists believed that the mind of anyone could be developed through correct instruction. De Condillac (1715-1789) thought that perception and sensation alone could be used to explain learning.

These ideas were of no help to the victims of colonial America. Deutsch (1949) reports that in the 17th and 18th centuries, the Puritans thought that anyone who was deviant was a witch or the victim of witchcraft. A common treatment

was burning or hanging. In New England, lunatics, distracted persons, people who were non compos mentis, and those who had fits were all grouped together, perhaps with vagabonds and paupers. Connecticut's first house of correction in 1722 was for: rogues, vagabonds, the idle, beggars, fortune tellers, diviners, musicians, runaways, drunkards, prostitutes, pilferers, brawlers, and the mentally afflicted. It was common practice to jail the mentally retarded or indenture them to citizens who offered to take responsibility for them for the lowest bid.

As late as 1792, Fodere published a treatise in which he speculated that cretinism occurred in places where the drinking water, or air, or something else was bad. These speculations were little advanced from those of Paracelsus in the 16th century. A cure for cretinism would have to wait until the next century. The search was spurred by concern with the spread of endemic cretinism.

In the latter part of the 18th century, more than the French and American revolutions were occurring. In 1792 Pinel ordered the chains to be taken off the residents of the Bicetre. This was so successful that Pinel ordered the same for the Salpetriere, another institution. Tuke in 1792 petitioned the Society of Friends to establish a mental hospital. A humane residential program for the mentally ill and mentally retarded was the outcome. This revolution resulted in a new attitude based on the idealistic hope that the mentally retarded could achieve normal functioning with humane treatment. The term moral management was coined, which was characterized by small-group community settings providing calm retreats for disturbed persons, systems of humane vigilance, elimination of physical abuse and chains, and freedom from indignities from staff members. Humanitarian reform movements were to affect the treatment of the mentally retarded through the first part of the 19th century.

The Dawn Of Special Education

A number of authors attribute the beginning of special education to a French physician named Itard for his work with a young boy who has become known as the Wild Boy of Aveyron or Victor. Although a number of individuals had worked with handicapped and gifted children probably from the beginning of time, Itard's work has been cited as the first documented, systematic approach to providing an education to a mentally retarded boy. The important issues that surround the case of Victor are intimately associated with the boy's care before he received treatment from Itard, the philosophical and theoretical views of Itard, and finally, the outcome of treatment.

Reports by peasants from the Lacune region of France about a naked boy in the woods searching for acorns and roots

began surfacing in 1797. In 1798, Victor was captured by woodsmen and put on public display in the village of Lacune. He eventually escaped into the woods. This encounter between the boy and the society of his time points to several of the issues that are problematic to a system which is not designed to deal with individuals who are different. In this particular case, once the curiosity of the townspeople of Lacune was satisfied by the public display of the wild boy, what else was there to be done? As soon as surveillance of the boy became lax, he escaped. What should have been done? Who should have done it? Why should they have done it? If the case of Victor were viewed from a contemporary position, it could be argued that the society was responsible for Victor and should have provided him with some measure of education so that he could become a participating member of the society.

Later, in January of 1800, the Wild Boy was spotted in Saint-Sernin. This time, after his capture, Victor was sent to the orphanage at Saint-Affrique and many agencies and individuals became involved with the boy. Victor was examined by a number of physicians, including Pinel, who diagnosed Victor as suffering from idiocy and insanity. Pinel felt that there was no hope for improving Victor's condition.

Itard, a former student of Pinel's, disagreed and felt that Victor's deficiencies were due to a lack of appropriate sensory experiences. It should be noted that Itard was greatly influenced by the writings of de Condillac, who wrote that people could become highly rational solely on the basis of sensory experience. Itard, guided by the philosophies of Locke and de Condillac, began his training program for Victor. The goals for Victor were:

1. To interest him in social life by rendering it more pleasant to him than the one he was just leaving, and above all more like the life which he had just left.
2. To awaken his nervous sensibility by the most energetic stimulation, and occasionally by intense emotion.
3. To extend the range of his ideas by giving him new needs and by increasing his social contact.
4. To lead him to the use of speech by inducing the exercise of imitation through the imperious law of necessity.
5. To make him exercise the simplest mental operations upon the objects of his physical needs over a period of time, afterwards inducing the application of these mental processes to the objects of instruction. (in Scheerenberger, 1983, p. 76)

In essence, Itard's goal was to make Victor **normal**. Itard's optimism represented a clear break from the former

position that the mentally retarded are hopeless creatures. The optimistic start of Victor's training led to a major disappointment as Itard came to the realization that Victor was not **cured** of his retardation.

Although the educational experiment with Victor did not produce the results which were expected, Itard in his report to the Minister of the Interior placed Victor's progress in perspective. "If one limits oneself to the two terms of comparison offered by the past and present states of young Victor, one is astonished at the immense space which separates them; and one can question whether Victor is not more unlike the 'Wild Boy of Aveyron' arriving at Paris, than he is unlike other individuals of his same age and species" (Scheerenberger, 1983, p.78).

This period in the history of mental retardation sharpens the focus on the question of what can be done with people who are mentally retarded. Is change possible? If so, how much change can be expected? Are techniques available to make the change possible? Does the society have the will to pursue the education of the mentally retarded?

Following his work with Victor, Itard took on Edourd Seguin as a student. Seguin not only continued Itard's work, but became a highly influential figure in the field of mental retardation. Mental retardation, Seguin thought, was the result of a deficiency in man's prominent expressions, i.e., activity, intelligence, and will. One of his major contributions to the field was the development of the physiological method of treatment for mental retardation. This method was presented in his text, Idiocy and Its Treatment by the Physiological Method. The method included training the muscular system, training the nervous system, educating the senses, acquiring general ideas, developing the ability to think in abstract terms, and acquiring a strong understanding and practice of moral (social) precepts.

Seguin used a number of methods and materials which he adapted from Itard and others. This period in the history of mental retardation reflected the emergence of such techniques as the use of positive reinforcement and modeling, which are still in use.

Maria Montessori (1870-1952) felt that retardation was a pedagogical rather than a medical problem and like Itard and Seguin, felt that the condition of the retarded could be improved through education. Her approach included the use of self-teaching didactic materials in a structured environment and sensory motor training. In the early 1900's, she established the first Casa Dei Bambini in Rome. Her reputation grew throughout the world, but it wasn't until the 1950's that her schools began to develop in the United States.

The Americans

While a number of events in Europe were occurring which would lead to changes in the treatment of the mentally retarded. Several important events were also occurring in the United States. Benjamin Rush (1745-1813), a physician, supervised the mentally ill and mentally retarded section of the Pennsylvania Hospital. Mental retardation, or **fatuity** as Rush termed it, was thought to be an absence of understanding and memory. Rush believed that the characteristics of the retarded varied widely. His contribution to the field was probably in bringing attention to the condition. Although he was a contemporary of Itard, he did not advocate any treatment for the mentally retarded.

Samuel Gridley Howe (1801-1876), another physician, became involved with the mentally retarded through his work as the director of an asylum for the blind in Massachusetts. His program became the Perkin's Institute and Massachusetts School for the Blind. In 1839, a blind student who was also mentally retarded was accepted into the institute. Howe's success with this student and others prompted him to work towards establishing a facility for the mentally retarded. As a result of his work, a commission was formed to study the problem, and, in 1848, a wing of the Perkin's School was opened, on an experimental basis, to provide an education to ten mentally retarded children. The experiment was a success, and, in 1855, the Massachusetts School for Idiots and Feeble-Minded Youths was established in Boston.

Howe did not achieve his successes alone. Charles Sumner (1811-1874) and Horace Mann (1796-1859) were instrumental in providing the impetus for change in the treatment of the retarded. Sumner's brother, George, also played a key role during this time. He visited Seguin's school in France and provided reports which were often quoted by Howe in his work.

Another major influence during this period was Dorothea Dix (1802-1877). While visiting jails, alms houses, poorhouses, and mental hospitals, she observed the treatment which people who were labeled insane and mentally retarded received. They were confined in cages, in rooms which lacked heat in winter, in cellars without lighting, and in a number of other undesirable accommodations. Dix saw people in chains, people dressed in rags or wearing nothing at all, people being beaten to control their behavior, and people tearing at their own skin for unknown reasons. Her testimony before the Massachusetts legislature in 1843 about these conditions is a moving account of the degradation which can befall the mentally retarded and others who find themselves institutionalized. This movement to make institutions more humane would be followed by a number of other advances in the treatment of individuals who are considered mentally retarded or otherwise different (Rosen, Clark, and Kivitz, 1976).

The latter part of the nineteenth century was a time in which a concern with heredity and its effect on human behavior became a dominant theme. A number of individuals became involved in studying paupers, mental defectives, and others who were considered to be undesirable elements in the community. Sir Francis Galton wrote of the dangers of allowing the "unfit" to propagate (Kanner, 1964). In the United States, Richard L. Dugdale published a survey titled "The Jukes, A Study in Crime, Pauperism, Disease, and Heredity." This survey traced a family identified as the "Jukes" across several generations. Dugdale obtained information on about 700 of the approximately 1200 descendants of this family. He found that 140 had been imprisoned for crimes, 280 were paupers who depended on public support for existence, and that the majority were of low physical and moral standards (Kanner, 1964). A number of others also investigated genetic factors during this period.

Early in the twentieth century, Henry Goddard wrote of the "Kallikak" family. Kanner (1964) states that Goddard made up the name Kallikak by combining two Greek words: kalos, meaning attractive and pleasing and kakos, meaning bad and evil to denote the two sides of the family he was studying. Goddard's study followed two branches of the Martin Kallikak family. Early in the American Revolution, Martin Kallikak met a feebleminded girl in a tavern and became the father of Martin, Jr., who was to be the first in a long line of people whom Goddard labeled as feebleminded, immoral, and criminal. A second branch of the family was started after Martin Kallikak left the army and married a respectable girl. Descendants of this branch, according to Goddard, became respectable citizens. He concluded that feeblemindedness is hereditary and that it should be controlled by segregation and colonization (Kanner, 1964). This study was used by individuals trying to reduce the number of mentally retarded people. Kanner (1964) reports on a meeting of the American Breeder's Association in which a number of solutions to the problem of mental retardation were discussed. Included among these suggestions were such things as sterilization, euthanasia, restrictive marriage laws, scientific breeding, and institutionalization. By 1926, sterilization laws had been enacted in 23 States.

While the movement towards defining mental retardation as a genetically transmitted condition developed, another movement was emerging from the work of Binet and Simon in France. They developed a scale that would help predict school success. Goddard, in addition to his work on the Kallikak family, also became involved in test development. In 1911, he translated the Binet-Simon scales into English (Patton, Payne, and Beirne-Smith, 1986). A few years later, Terman at Stanford refined the scales into what has become the Stanford-Binet, which is still widely used as a measure of intellectual functioning. The development of the testing

movement provided a method for categorizing mental retardation along a continuum of intellectual functioning. Although the use of tests such as the Stanford-Binet has been questioned in terms of whether intellectual assessment is biased, these tests have provided a common metric on which to base decisions about a person's intellectual capacity.

Although intelligence tests had a tremendous impact on the field of mental retardation, another major influence on the field has been the development of instruments to measure adaptive behavior. Mental retardation was viewed from earliest times as a condition in which the individual has a difficult time adjusting to the demands of his or her socio-cultural environment. This view led a number of individuals to propose a social competence view of mental retardation. Edgar Doll, a proponent of the social competence position, proposed a definition in which social competence was the primary component of mental retardation. Additionally, he developed an instrument, the Vineland Social Maturity Scale, to assess an individual's social competence (Farber, 1968).

The period between the 1930's and the 1950's was a relatively quiet time in the field of mental retardation. Recovery from the Depression and World War II became a primary preoccupation of the American society.

The 1950's began an era in which mental retardation again became an important social issue. In 1950, the National Association for Retarded Children was formed to respond to the dearth of services available to mentally retarded children by forming private agencies and seeking legal remedies. By the mid 1950's, the majority of States had laws making education available for mentally retarded students. These laws, however, often applied only to the mildly retarded. The parents of the moderately and severely retarded students were still forced to seek services in private agencies. Educators were also becoming concerned about segregation of the mentally retarded. Those who received services often received them in self-contained classrooms or special schools away from the mainstream of American education.

In 1959, the American Association on Mental Deficiency proposed a new definition of mental retardation which included concepts of impairment in maturation, learning, and social adjustment. The practice of using an IQ score as the sole determiner of mental retardation was virtually unaffected by this definition.

Resolution of the various conflicts would have to wait until the next two decades. In 1961, President John F. Kennedy established the President's Panel on Mental Retardation to help guide national policy aimed at the mentally retarded. They presented their final report, making 112 recommendations on issues related to research, prevention, provision of services, legal and social concepts, and public awareness (Scheerenberger, 1983). President

Kennedy, after review of the recommendations, addressed the Congress, seeking support for recommendations presented by the panel. The response by the Congress can be gauged by the fact that, over the next twenty years, 116 acts or amendments provided support for the mentally retarded.

In education, the 1960's represented a time of introspection. Questions were being raised about the efficacy of special programs for the mentally retarded. G. Orville Johnson and Lloyd Dunn, among others, questioned the efficacy of special programs that, in their opinion, did not prepare mentally retarded students any better than regular programs. Questions of special class placement, coupled with concerns about labeling and classification, and a renewed interest in civil rights created an environment in which "mainstreaming" was the object of focus (Scheerenberger, 1983).

Kolstoe (1976), in referring to the work of Johnson and Dunn, added to the controversy by agreeing that, in the traditional academic areas, mentally retarded students in special programs were not doing any better than students who were not in special programs. He pointed out, additionally, that the role of the special class was not only to provide academic instruction, but also to provide the kind of instruction that would lead to eventual adult adjustment. Kolstoe presented evidence that mentally retarded students in work-oriented special classes were more successful in the area of employment than those who had not received the training.

The positions for and against special class placement taken during the 1960's continued into the 1970's. A number of landmark cases, including one brought by the Pennsylvania Association of Retarded Children against the Commonwealth of Pennsylvania, began to speak to the issues of providing services to the mentally retarded. In general terms, the courts found that mentally retarded individuals had rights to education and other services.

Public Law 94-142, the Education for All Handicapped Children Act of 1975, was passed to insure that all handicapped children would be provided with a free and appropriate education in the least restrictive environment. This law addressed the questions raised in the 1960's about placement of students into special classes and the appropriateness of the education received. The 1970's were a time of intense legislative and litigative action that moved services for the mentally retarded to a new level.

A number of events in the 1980's had a significant effect on the field of mental retardation. Educational programming at birth for handicapped children was mandated by P.L. 99-457, the Education of the Handicapped Act Amendments of 1986. In 1990, P.L. 101-476, known as the Education of the Handicapped Act Amendments and also as the Individuals with Disabilities Education Act (IDEA) mandated transition

services to all students with disabilities. IDEA also changed the term handicapped to disability to reflect a more positive focus on what individuals can do. Proponents of expanded educational opportunities point out the necessity of providing greater services to ensure equity for the mentally retarded. At this time, educational services are provided for the mentally retarded from birth through their 21st birthdays. Prohibition of discrimination on the basis of disability in private sector employment, public services, public accommodations, transportation, and telecommunications was established by P.L. 101-336, the Americans with Disabilities Act of 1990.

DEFINITION

The definition of mental retardation has depended on the needs and attitudes of the social system within which it was defined. In preindustrial revolution societies, physical prowess and manual labor were prized more than the ability to read. The majority of people were not literate, but lack of academic skills did not necessarily have an adverse impact on one's chances for survival. To define someone as handicapped meant that the person did not meet the rudimentary physical and mental demands of the times. The earliest definitions of mental retardation relied more on judgments of social behavior rather than on scientifically based test data.

In 1845, Esquirol proposed a definition for idiocy based on intellectual faculties as manifested in one's knowledge compared to others of the same age and experience:

> Idiocy is not a disease, but a condition in which the intellectual faculties are never manifested, or have never been developed sufficiently to enable the idiot to acquire such amount of knowledge as persons of his own age and placed in similar circumstances with himself are capable of receiving.

Idiots were distinguished from imbeciles by the degree of speech development they manifested. Today, language development is still often used as a criterion to separate the severely retarded from the profoundly retarded because IQ tests are generally much less accurate for these groups.

In the second half of the nineteenth century, definitions were influenced by the optimistic viewpoint that mental retardation could be cured. Seguin was a sensationalist who hypothesized that the brain could atrophy through disuse and that, therefore, mental retardation could be associated with mental inactivity. The cure he proposed was to stimulate the brain through sensory experiences. This optimistic attitude was gradually replaced by the realization that central nervous system (CNS) damage was not curable and that mental retardation was pathological in the cases of idiots and imbeciles.

As societies became more technologically advanced, their needs for individuals with greater intellectual skills increased. It was necessary for many individuals in the workforce to acquire skills more academic in nature in order to simply maintain their jobs. As literacy became more valued, those who could not satisfactorily demonstrate academic skills became marked. In fact, over 90% of those called retarded today would have been classified as normal in preindustrial revolution societies.

The demand for increased academic competency was not the only factor to influence the definition of mental retardation. A parallel development in the sophistication of testing also influenced the definition to a great degree. As practitioners have felt more comfortable with testing, cut-off scores have increasingly found their way into the operational criteria of definitions.

The development of the Binet-Simon Scales was a major step in the measurement of intelligence. Although the construct of intelligence had been defined in a number of ways, Binet based the development of the 1905 scale on the premise that a wide range of functions, including judgment, comprehension, and reasoning, constituted intelligence (Matarazzo, 1972). In a report in 1908, Binet developed and formally introduced the concept of intelligence based on mental age. A child's mental age was determined on the basis of the number of items passed on the Binet-Simon Scales. In 1910, Goddard defined mental retardation based exclusively on mental age scores. Imbeciles were defined as those whose mental age was under three. Idiots had mental ages from three to seven. The apparent precision of mental age allowed Goddard to include a third level in his classification system. Morons were defined as those whose mental age was between eight and twelve.

As time passed, the policy of relying exclusively on test scores to define mental retardation was questioned. Tredgold in 1937 defined mental retardation in terms of social/behavioral factors. He stated that mental retardation was:

> a state of incomplete mental development of such a kind and degree that the individual is incapable of adapting himself to the normal environment of his fellows in such a way as to maintain existence independently of supervision, control, or external support.

This social incompetence definition sparked a debate which was not resolved until experts constructed definitions which incorporated both low IQ scores and social incompetence.

Besides increased academic competencies and greater test sophistication, a third factor influencing definition is the conception that mental retardation is a multidimensional handicap. Instead of relying exclusively on low mental age,

a low intelligence quotient, poor language development, or social incompetence, the definition has evolved to reflect difficulty in functioning in the normal environment due to mental subnormality.

In 1941 Doll proposed a definition based on six criteria:

> The mentally deficient person is (1) socially incompetent, that is, socially inadequate and occupationally incompetent and unable to manage his own affairs; (2) mentally subnormal; (3) retarded intellectually from birth or early age; (4) retarded at maturity; (5) mentally deficient as a result of constitutional origin, through heredity or disease, and (6) essentially incurable.

Refinements of this and other definitions continued until Heber revised his 1959 definition in 1961. This definition became accepted by a majority of those who worked with mentally retarded children at that time. Heber's definition is as follows:

> Mental retardation refers to (1) subaverage general intellectual functioning, (2) which originates during the developmental period, and (3) is associated with impairments in adaptive behavior.

Heber defined subaverage general intellectual functioning as a score on a standardized intelligence test which is greater than one standard deviation below the mean. This included approximately one-sixth of the population. The developmental period, according to Heber, includes the first sixteen years of life. Impairments in adaptive behavior were defined differently at different age levels. In the preschool years, difficulty is indicated in delayed walking, talking, and other developmental skills. During the school-age years, impaired learning ability was thought to be indicative of nonadaptive behavior. As adults, mentally retarded people exhibit poor social adaptation as seen in lack of independent living skills and failure to obtain and hold a job in a competitive employment situation.

The most often used definition today is one developed by Grossman. In the current American Association on Mental Deficiency (AAMD) manual, Grossman's (1983) revised definition refers to mental retardation as:

> significantly subaverage general intellectual functioning existing concurrently with deficits in adaptive behavior and manifested during the developmental period.

Significantly subaverage general intellectual functioning is evidenced by a score on a standardized intelligence test which is greater than two standard deviations below the mean. This IQ range represents less than three percent of the population. Deficits in adaptive behavior are characterized

as difficulties in maturation, learning, independence, and/or social responsibility as observed in the student's home, school, and neighborhood environments. The developmental period, according to Grossman, is defined as a period between conception and age eighteen.

CLASSIFICATION

Classification, in theory, facilitates delivery of services by providing a rational basis for grouping and an indication of characteristics and functioning level of the individuals involved. In practice, IQ has been the most important diagnostic element in mental retardation. IQ was designed to predict failure in school so that those who could not profit sufficiently from regular education could be handled in differential fashion. Once a cut-off point was established, mentally retarded children were further classified, most commonly by degree of retardation (IQ), etiology (cause), or, as adults, by functioning level (life chances).

Degree

Scores on IQ tests are currently used to indicate the degree or level of mental retardation. Mild mental retardation is defined by IQ scores ranging approximately from 70 to 55. Other terms used to denote mild retardation include educable mentally retarded and moron.

Moderate mental retardation is defined by IQ scores ranging approximately from 54 to 40. Synonymous terms for moderate retardation include trainable mentally retarded and imbecile.

Severe mental retardation is defined by IQ scores ranging approximately from 39 to 25. Profound mental retardation is defined as an IQ score below 25. Both categories have been referred to as sub-trainable or custodial.

Etiology

Classification by etiology often produces elaborate systems. These systems may not provide educators with directly applicable information, but they are important in understanding the phenomenon of mental retardation. The American Association on Mental Deficiency (AAMD) currently uses a system including nine categories. The following discussion is based on those categories:

I. Infection or Intoxication

Infection is caused by an invasion of the body systems by bacteria, protozoa, viruses, or other parasites. Although

relatively rare, mental retardation may result if the central nervous system (CNS) is damaged. Six infectious agents are described.

A. Congenital cytomegalovirus is a prenatal salivary gland infection of the mother. Although there is no demonstrable illness in the mother, the fetus may be severely affected. Symptoms in the infant may include any, all, or none of the following: microcephaly, anemia, jaundice, enlarged liver or spleen, hydrocephaly, cerebral calcification, varying degrees of mental retardation, deafness, blindness, cerebral palsy, cataracts, and convulsive disorders. Of all the viral infections, cytomegalovirus is thought to be the one with the most likelihood of causing neurological damage in infants.

B. Rubella or German measles is a virus which can cross the placental barrier and attack the central nervous system of the fetus. Mental retardation is most likely to occur if the mother contracts the virus during the first trimester (first three months) of pregnancy. Microcephaly (small head size) and congenital cataracts may be present at birth. Heart disease and deafness are also possible. Gregg linked Rubella to congenital cataracts in 1941. Since then the devastating effects of this disease have been chronicled. A vaccine was licensed to combat the virus in 1969.

C. Syphilis is a venereal disease. Its bacterium crosses the placental barrier after the fifth month of pregnancy and may cause mental retardation, blindness, and deafness in the developing fetus. Abortions, miscarriages, and stillbirths are also caused by syphilis. There are early and late forms of the disease. The early form is marked by rashes, sniffles, moist lesions at mucocutaneous junctions, pseudoparalysis of limbs, and enlargement of the spleen, liver, and lymph nodes. Central nervous system involvement includes hydrocephalus, convulsions, and mental retardation. If left untreated, syphilis may cause a delayed attack on the CNS called juvenile paresis. Neurological symptoms, speech disturbances, and psychoticlike manifestations appear between ages 8-10 in boys and 10-12 in girls. The classic Hutchinson triad of this stage consists of nerve deafness, interstitial keratitis, and Hutchinsonian incisors. Mental deterioration is progressive.

D. Toxoplasmosis is a congenital or acquired protozoic infection. The organism may be carried in raw meat and cat feces. It is especially dangerous to the fetus if contracted during the first two trimesters of pregnancy, but may also be contracted postnatally. Congenital toxoplasmosis may be associated with a pathological condition of the choroid coat of the eye and retina, cerebral calcification, hydrocephaly, microcephaly, psychomotor disturbances, epileptic seizures, feeding difficulties, sudden rises in temperature, and increased muscle tone. About 85 percent of those who survive are mentally retarded, ranging from slight to profound.

Acquired toxoplasmosis may sometimes lead to encephalitis.

E. Encephalitis is a postnatal inflammation of the brain caused by any one of several infectious agents, including measles, scarlet fever (roseola), whooping cough, pneumonia, rubella, polio, varicella (chicken pox), and toxoplasmosis. Mental retardation may result if the fever is high and extends over several days. The effects of encephalitis may be immediate or delayed for several years.

F. Meningitis is an inflammation of the brain-lining membrane, which may occur during the prenatal (before birth), perinatal (during birth), and postnatal (after birth) periods. About four in 10,000 live births are affected and the survival rate is about fifty percent. Aseptic meningitis is usually caused by viruses. If it occurs in the prenatal period, there may be extreme CNS, respiratory, or circulatory involvement. Many of its victims are mentally retarded, have seizures, and have neuromotor disabilities. Bacterial meningitis may lead to mental retardation (mild to severe), hearing loss, seizures, neuromotor problems, and speech defects.

F. Genital herpes is a virus which can be acquired during delivery as the baby passes through the birth canal. It is associated with meningoencephalitis, microcephaly, and cerebral calcification. Delivery by cesarean section prevents acquisition of genital herpes by the baby.

Intoxication is the poisoning which occurs when an organism comes into contact with toxic agents. Toxicity varies according to the level ingested or absorbed. In great quantities, almost any agent can be toxic. The resultant central nervous system damage can cause mental retardation.

Chemical agents damage the CNS in two ways. The first is by depriving the brain of oxygen (anoxia). Cyanide, carbon monoxide, and severe barbiturate poisoning may cause anoxia and subsequent brain damage.

The second is by damage to specific nerve structures. Organomercury compounds, dissolved organic phosphates, hydrocarbons, herbicides, fertilizers, fungicides, phenols, pesticides, and sulfur compounds may attack the nervous system. Permanent damage may be the result of ingestion or absorption of chemical agents such as alcohol, toluene (glue), gasoline, methanol (wood alcohol), amphetamines, and other stimulants, depressants, and hallucinogens. The CNS may also be damaged by metals such as lead, arsenic, mercury, nickel, cobalt, manganese, iron, zinc, lithium, and selenium. If the resulting central nervous system damage is severe enough, mental retardation results.

Under special circumstances of blood group incompatibility between the mother and her fetus, antibodies produced in the mother's spleen will attack the red blood cells of the fetus. During this process, bilirubin is released. When not bound to red blood cells, bilirubin is

toxic to the CNS and may do enough damage to cause mental retardation. During pregnancy, the maternal liver metabolizes the bilirubin and protects the fetus. After the child is born, the immature liver is unable to metabolize the bilirubin and it builds to toxic levels. Blood is classified according to its components. One of these is a protein on the surface of red blood cells. Those who have this protein are said to be Rh-positive and comprise over 85 percent of the population; those who don't are classified as Rh-negative, determined by recessive gene transmission. If an Rh-positive child is conceived by an Rh-positive male and an Rh-negative female, antibodies develop in the female after the pregnancy is terminated by birth, miscarriage, or abortion. These antibodies will attack the CNS of the next Rh-positive fetus conceived. Fortunately there is a way to destroy the Rh-positive blood cells which remain in the mother's bloodstream after the pregnancy is terminated. Injection of Rh immunoglobulin (RhoGAM) shortly after the pregnancy is terminated makes it unnecessary for the mother's system to produce the antibodies because the foreign Rh-positive cells are eliminated. Rh-positive babies born with maternal antibodies in their system are treated by phototherapy. Blood transfusions may also be necessary.

Fetal alcohol syndrome may occur in up to seven per 1000 live births. By the 1970's, it was confirmed that maternal prenatal consumption of alcohol could produce a mentally retarded child. It is clear that moderate to heavy maternal drinking during pregnancy is also related to growth retardation, low brain weight, heart defects, facial anomalies, and orthopedic defects.

II. Trauma or Physical Agent

Physical trauma or damage by a physical agent may occur prenatally, perinatally, or postnatally. In the prenatal months, the fetus may suffer brain damage due to X-rays. During delivery, anoxia may be caused by a placenta which detaches too soon, kinking of the umbilical cord, and strangulation if the umbilical cord wraps too tightly around the baby. Postnatally, blows to the head, penetrations of bone or foreign objects into the brain, and secondary damage caused by cerebral edema (swelling), hematoma (accumulations of blood), and thrombosis (clots or obstruction) may cause brain damage.

Two main causes of brain damage are accidents and child abuse. In many instances, the resulting brain damage, which could have been prevented, causes mental retardation.

III. Metabolism Or Nutrition

Disorders in this category account for about five percent of those classified as mentally retarded. Metabolism

and nutrition overlap in the areas of digestion, absorption, transportation, and utilization of nutrients. There are six classes of nutrients: proteins, carbohydrates, lipids, vitamins, minerals, and water (Hamilton and Whitney, 1990). Imbalances related to amino acids, carbohydrates, and lipids as well as endocrine disorders and nutritional deficiencies can lead to mental retardation.

A. Lipid Disorders

Lipids, the general term for fats (solid at room temperature) and oils (liquid at room temperature), are composed of the triglycerides (which comprise 95 percent of the lipids found in food), phospholipids (including lecithin), and sterols (including cholesterol). Excess calories are stored by the body as triglycerides. The origin (proteins, carbohydrates, or lipids) of this fat is not particularly meaningful, since any of the energy nutrients can be stored as triglycerides. Fat is used to pad all vital organs, carry fat-soluble nutrients, help maintain body temperature, pad cells, and serve to supply essential fatty acids (Stare and McWilliams, 1984). Lipid disorders involve either abnormal fat metabolism due to a faulty enzyme or the deposit of fatty substances in the ganglion cells of the CNS, the peripheral nerves, or the skin and linings of the internal cavities of the body, e.g., blood vessels and heart. These disorders are usually progressive and degenerative, with onset ranging from the first year of life into the adult years.

Amaurotic Familial Idiocy is manifested in four forms. The infantile form is called Tay-Sachs disease or Sphingolipidose. It is more prevalent in Ashkenazi Jews, a group from eastern Europe. About one in 30 Ashkenazi Jews, one in 100 Sephardic Jews, and one in 300 Yeminite Jews and non-Jews are carriers. This autosomal recessive genetic defect causes improper lipid metabolism, degeneration of the brain, progressive deterioration of nervous tissue, and death. Symptoms include hyperactivity, unusual sensitivity to light and sound, weakness, apathy, difficulty holding up the head, regression of the grasping reflex, convulsions, gradual deterioration of vision, spastic paralysis, cranial enlargement, severe mental retardation, and a deterioration of the macular area of the retina which is manifested by a cherry-red spot at the site. Onset is usually at three to six months and death occurs from one to three years after onset. At death, victims show enlarged CNS ganglion cells and increased neuraminic acid in the cerebral cortex.

The late infantile form is called Bielchowsky-Jansky disease. It is more common in non-Jewish families than the infantile form. The symptoms are similar to Tay-Sachs, but the onset occurs at two to four years and the deterioration occurs at a slower rate.

The juvenile form is called Batten-Spielmeyer-Vogt disease. It is rare in Jewish children. Onset is from ages three to ten. Symptoms include blindness, impaired balance and coordination, convulsions, mental deterioration, and psychotic-like behavior.

The late juvenile form is Kuf's disease. It's onset is from 15-25 years. Symptoms are the same, but deterioration is relatively slow.

Gaucher's Disease or cerebroside lipidosis occurs in an acute form and a chronic form. In the acute form, there is enlargement of the liver, spleen, and lymph nodes caused by deposits of cerebrosides usually found only in the brain. Regression in development level, general physical deterioration, spasticity, general apathy, and lack of response characterize the acute form. Onset is at the age of four to five months and the disease is fatal. The chronic form has its onset in later childhood and usually the nervous system and intelligence are not affected.

Niemann-Pick Disease or sphingomyelin lipidosis is similar to Tay-Sachs. Transmission is by a defective autosomal recessive gene which causes retention of sphingomyelin in the liver, spleen, and lymph glands, resulting in their enlargement. Victims are characterized by weight loss, dehydration, and progressive paralysis. Visual deterioration with the characteristic cherry-red spot and deafness may also be exhibited. The onset occurs in infancy and death occurs early.

Mucopolysaccharidosis is also known as gargoylism or lipochondrodystrophy. A mucopolysaccharide is deposited in the body, with highest concentrations in the liver, heart, lungs, spleen, and brain. By the age of two, mental retardation is usually evident and may range from mild to severe. Most organs are affected and the physically normal infant is transformed into a grossly deformed child. The most common form is known as Hurler's syndrome or Mucopolysaccharidosis Type I. It is inherited by autosomal recessive gene transmission and the victim is prone to dwarfism. Other symptoms include an enlarged head, short neck, limited movement in the joints, bushy eyebrows, saddle-shaped nose, thick lips, a protruding tongue, and cloudy corneas. Death usually results before the victim reaches the pre-teen years. A less common form is Hunter's syndrome or Mucopolysaccharidosis Type II and is transmitted by a sex-linked recessive gene. Symptoms are similar to Type I, except that the corneal clouding is not present and mental retardation is less severe. The victim is more likely to be affected by nerve deafness and may live into the middle years. One other form is associated with mental retardation. The Sanfilippo syndrome or Mucopolysaccharidosis Type III results in severe mental retardation. Mucopolysaccharidoses are differentiated according to the specific enzyme defect responsible for the symptoms.

B. Carbohydrate Disorders

Carbohydrates are composed of sugars, starches, and fibers. Of the energy nutrients (proteins, carbohydrates, and lipids), carbohydrates are the most ideal source of fuel (Hamilton and Whitney, 1990), preferentially used by the CNS. The complex carbohydrates (starch and fiber) and the naturally occurring sugars (glucose, fructose, and mannose) found in fruits, vegetables, and milk are more beneficial than the concentrated sweets, e.g., brown or white sugars, syrups, jellies, and honey.

Galactosemia is an example of a carbohydrate disorder caused by recessive gene transmission. In the most common form (of two), the body is unable to metabolize galactose, a sugar found in milk, because of a lack of galactose-1-phosphate uridyl transferase. Symptoms include jaundice, weight loss due to frequent vomiting, cataracts, and various degrees of IQ loss. If the disease is discovered early enough, the symptoms disappear and mental retardation is prevented. Treatment consists of removing milk from the diet.

Fructosemia or hereditary fructose intolerance occurs when the enzyme hepatic fructokinase used to metabolize fructose (the sugar contained in fruit) is absent. Ingestion of fruit causes a sharp drop in the blood glucose level. Symptoms include a general appearance of malnutrition and mental retardation.

Idiopathic Hypoglycemia is a low, uncontrollable blood sugar level, which deprives the brain of energy. The condition is caused by glycogen storage disease, glycogen synthetase deficiency, hereditary fructose intolerance, or galactosemia. Symptoms first occur prior to the age of two. At first the child has seizures and stares. This is followed by fatigue, weakness, flushing, sweating, speech problems, and visual disturbances. Brain functioning is depressed and central nervous system anomalies may be present, resulting in mental retardation. Symptoms generally decrease with age. ACTH injections are beneficial in most instances.

C. Protein or Amino Acid Disorders

Proteins are nitrogen-containing substances which may be used for growth and maintenance of tissue and are important in the regulation of fluids, salts, and pH (acid-base) balance (Guthrie, 1986). Hemoglobin, antibodies, enzymes, and hormones have proteins as their major components. Proteins are built by combinations of twenty-two amino acids called alpha amino acids (Mason et al., 1973) and form the principal components of the cell protoplasm (Hamilton and Whitney, 1990). Several disorders in this area may cause mental retardation.

Phenylketonuria (PKU) is an autosomal endogenous

metabolic disorder resulting from defective recessive genes. About one in every 10,000 to 12,000 or so live births is affected. Both parents must be carriers. It is called a protein or amino acid disorder because an enzyme needed to metabolize the amino acid phenylalanine is absent or in short supply. Phenylalanine is a common amino acid found in milk and other substances. Ordinarily the enzyme called hepatic phenylalanine hydroxylase metabolizes phenylalanine into tyrosine. In PKU, hepatic phenylalanine hydroxylase is absent or in short supply. As a result, phenylalanine is not completely metabolized. The incomplete metabolites formed include phenylpyruvic acid and phenylketones, both of which are toxic to the CNS and cause damage when allowed to build to critical levels.

PKU was discovered in 1934 by Folling. Babies who test positive for PKU during routine postnatal screening are placed on a diet low in phenylalanine, usually soybean based. CNS damage does not ordinarily occur after the age of five or so due to normal changes in metabolic processes. PKU children, however, are often kept on special diets to help them overcome behavior disorders. IQ levels may range from normal to profound mental retardation depending on how early the diet was started and how faithfully it was followed. It is possible for a non-PKU fetus to suffer CNS damage if the mother is afflicted with PKU. In that case, a special diet is prescribed for the mother which reduces the levels of phenylalanine and ketones in the intrauterine environment.

Hartnup Syndrome is also an endogenous metabolic disorder which is extremely rare. It is caused by defective recessive genes. Tryptophen is incorrectly metabolized and the body excretes excessive amounts of amino acids and indole derivatives. Symptoms include a pellagra-like skin rash, hypersensitivity to light, cerebellar ataxia, and, in most cases, mental retardation. Treatment with Vitamin B has been helpful in some cases.

Wilson's Disease or Hepatolenticular Degeneration is transmitted by an autosomal recessive gene. It is characterized by copper deposits in the brain, liver, and other organs. Active onset of symptoms is delayed until the individual is 20-30 years old and death results within ten years. Symptoms include tremors, muscle spasms, rigidity, convulsions, parkinsonism, torticollis (twisting of the neck), mental retardation, and poor speech development. This condition is extremely rare. Treatment has centered on the use of substances which will promote the secretion of copper.

Maple-Syrup Urine Disease was named for the characteristic odor of the urine of its victims. Onset is in the perinatal period. Metabolism of the branched chain amino acids (leucine, isoleucine, and valine) is defective. Treatment involves providing a diet low in those amino acids. Severe mental retardation and neurological disorders often result.

Lowe's Disease is also known as Cerebro-ocular-renal Disease. It is rare, caused by defective sex-linked recessive genes. Reported in 1952, the result is severe mental retardation. Other symptoms include acidosis, excessive amino acids in the urine, glaucoma, cataracts, and spongy bones with rickets.

D. Endocrine Disorders

Hypothyroidism is divided into congenital and acquired forms. Cretinism is a congenital disease, caused by a severe deficiency or complete lack of the thyroid hormone due to an absent or partially absent thyroid gland. At least three of the inherited types are transmitted by a defective recessive gene which interferes with the enzyme system. Goiter (an enlargement of the thyroid gland) is usually present in the inherited versions in which at least some part of the thyroid gland is present. Other characteristics include a large head, flat and broad nose, prominent cheekbones, a characteristic eye fissure (straight, narrow, and horizontal), wide-set eyes, heavy eyelids, a wrinkled forehead, a large abdomen, shortened extremities, and decreased muscle tone. Mental retardation is common. Physical symptoms can be greatly reversed with thyroxin. The most common form is not inherited and is caused by an iodine deficiency or the presence of antibodies in the mother.

Hypoparathyroidism has inherited and non-inherited versions, but not genetic. Defects in the parathyroid function lead to lesions in the CNS, short stature, stubby hands, unusually round face, tetany (intermittent spasms), convulsions, cerebral calcification, cerebral accumulations of fluid, cataracts, psychotic-like behavior, and mental retardation.

Lesch-Nyhan disorder was described in 1964 and is also called congenital hyperuricemia. It is present in one out of 100,000 births. There is excessive uric acid in the blood due to deficient activity or nonexistence of the enzyme hypoxanthine guanine phosphorybosl transferase (HGPRT). This disorder is inherited through sex-linked recessive gene transmission and is manifested in an uncontrollable urge to cause injury to self and others, including the ripping and biting of one's own flesh. Victims have been known to bite off their own fingers. Mental retardation, gout, cerebral palsy, and normal pain sensations are present. When unrestrained, the victims scream, seemingly in fear of the pain they will inflict on themselves. Restraint most often has a calming effect.

Nephrogenic Diabetes Insipidus is a sex-linked recessive gene disorder characterized by the failure of the kidneys to respond to the antidiuretic hormone pitressin. The body can't hold fluids and the resulting prolonged dehydration leads to mental retardation. It occurs mostly in males.

Other symptoms include excessive thirst, vomiting, and erratic high fevers.

Prader-Willi syndrome is characterized by an insatiable appetite, with obesity as the consequence. Motor and mental development is retarded. Victims have small stature and features, including genitalia.

E. Nutritional Deficiencies

Animal studies show that calorie deprivation during the critical period of brain growth causes lower brain weight, a reduced number of central nervous system cells, smaller cell size, a reduced amount of DNA within cells, and less myelin (used to insulate nerve cells). Many of these anomalies are irreversible.

In humans, hemorrhage during pregnancy, toxemia, and prematurity are factors associated with mentally retarded offspring. Prematurity is the reproductive factor with the strongest known relationship to brain dysfunction. Poor diet causes a higher rate of infant mortality, prematurity, and birth defects among the disadvantaged than any other single cause. Malnutrition during both the prenatal and postnatal critical brain growth period can reduce the number of brain cells by as much as forty to sixty percent. Evidence indicates that the performance of individuals who suffered malnourishment in the critical brain growth periods never catch up to the levels of their well nourished peers.

IV. Gross Brain Disease

Although the symptoms of skin lesions and tumors most often appear postnatally, it is thought that syndromes in this category are hereditary, resulting from mutant or pathological genes. The primary syndromes are called neurocutaneous dysplasias or congenital ectodermoses. During development, the outer layer of the embryo (the ectoderm) supplies protoplasm to form the skin, nervous system, eyes, tooth enamel, and lining of part of the mouth, nose, and anus. Disorders in this process lead to CNS damage. Secondary syndromes involve degeneration of specific fiber tracts or neural groups.

It is estimated that gross brain disease causes about one half of one percent of mental retardation. The first three syndromes have been identified as types of congenital ectodermosis. The last, Huntington's chorea, is a degenerative condition of specific fiber tracts or neural groups.

A. Neurofibromatosis is also known as neuroblastomatosis or Von Recklinghausen's disease. It is chronic, progressive, and transmitted by a dominant autosomal gene. Only ten percent of those afflicted develop mental retardation (mild to severe) and epilepsy due to tumors

within the skull. The disease is characterized by light brown skin patches (cafe au lait) which appear at birth, skin tumors (neurofibromas) which appear in late childhood, and tumors on the nerves. It was discovered in 1882. About one in 3000 is afflicted.

B. Cerebral capillary angiomatosis is also known as nevoid amentia, trigeminal cerebral angiomatosis, encephalofacial angiomatosis, encephalotrigeminal angiomatosis, and Sturge-Weber-Dimitri Disease. Transmission is probably by a dominant autosomal gene. There is a port wine colored mark on the face in the area of the trigeminal nerve. Vascular malformations in the cerebral cortex probably account for the mental retardation, hemiplegia, and convulsive disorders associated with this disease. Mental retardation is found in varying degrees in approximately sixty percent of those affected.

C. Tuberous sclerosis is also known as Bourneville's disease or epiloia. About one in 20,000 to 40,000 is affected by this progressive disease which was recognized in 1880. It is transmitted by a dominant autosomal gene. In the classic form, epilepsy begins between the ages of three and six. Between the ages of five and thirteen, the child develops hardened areas of the cortex and a butterfly pattern rash of nodules on the face and back (adenoma sebaceum). There is progressive mental deterioration. Tumors may form in internal organs such as the kidneys and heart and on the CNS. Treatment consists of administering anticonvulsant medication to control seizures and surgery to remove tumors. Death occurs early, usually before the age of twenty-five. In the modified form of the disease, an independent pair of genes modify the syndrome so that there is normal intelligence, no health problems, and a normal life span.

D. Hunington's chorea. Transmission is by dominant gene, which occurs in about one in 10,000 births. This degenerative disease attacks nerves and fibers in the brain. At first, the victim exhibits emotional disturbance, tics, choreiform (jerky) movements, seizures, or a progressive rigidity without definite involuntary movements. As the disease progresses, dementia and choreic movements become more severe until death. Onset is usually about age forty. Because offspring may inherit the dominant gene before the parent exhibits the symptoms, it represents a unique genetic problem.

V. Unknown Prenatal Influence

This group of syndromes includes all skull abnormalities present at or prior to birth which have an unknown etiology and cause mental retardation. Craniofacial anomalies are discussed.

A. Cornelia de Lange syndrome. Marked by severe mental retardation, it is thought that this condition is transmitted

by an autosomal recessive gene. Identified in 1933, the syndrome is thought to affect one in 30,000 to 50,000 births. Typical symptoms include small stature, microbrachycephalic (small and flattened at the back) head shape, a wide upper lip, an upturned nose, and hairiness with confluent eyebrows and hair that grows low on the forehead and neck.

B. Microcephaly. It is thought that this insufficient skull growth is caused by a single autosomal recessive gene or prenatal factors such as exposure to excessive radiation from x-rays. The small, conical-shaped skull causes the CNS to be compressed. Mental retardation is severe to profound. Physical deformities include a curved spine and stooping posture which results in a monkey-like gait. The microcephalic child may exhibit uncontrollable behavior, seizures, spasticity, and hyperactivity.

C. Macroencephaly. The most common cause of an enlarged head is hydrocephaly. When spinal fluid is not drained off at a proper rate or when an excessive amount of spinal fluid is produced by an overacting choroid plexus, the resulting pressure may lead to progressive enlargement of the cranial vault in children in which the cranial sutures have not fused. Intelligence may range from normal to severe mental retardation, with early death quite common in untreated cases. Seizures are common. Hydrocephalus is related to maternal age. Less than one in 1000 mothers aged 25-35 give birth to hydrocephalic babies. After the age of 40, the incidence rises to two-three per 1000. Fortunately, pressure may be relieved by installation of a shunt system which drains the excess fluid from the brain into a vein in the neck. Drainage is controlled by a small valve (called a Holter valve) installed in the shunt tube. The whole system is placed beneath the skin and must be checked periodically for blockages.

D. Acrocephalosyndactylia is also called Apert's syndrome. In some cases, this is caused by a defective dominant gene. Characteristics include acrocephaly (a tower or steeple- shaped skull), narrow forehead, prominent eyes widely set and slanting downward to the outside, atrophication of the optic nerve, webbed hands and feet, and skeletal deformation. Skull deformity results from the skull's unusual response to normal pressure and the premature closing of the coronal suture.

E. Laurence-Moon-Biedl syndrome. This is transmitted by an autosomal recessive gene. Besides mental retardation, the syndrome is characterized by obesity, hypogenitalism, cerebral palsy, and retinitis pigmentosa. It was discovered in 1866. There is some debate about the differentiation of Laurence-Moon-Biedl syndrome and Bardet-Beedl syndrome. In Bardet-Beedl syndrome, cerebral palsy is not present, but those affected demonstrate polydactyly (extra digits).

VI Chromosomal Abnormality

The uniqueness of an individual is a result of the interaction of a number of factors, including those which are inherited. Children tend to manifest traits which have been passed on to them by their parents. Each parent contributes to the genetic inheritance of the child. The specific genes that a parent passes on to the child and the interaction of these genes with the genes of the other parent in large measure provide the developmental blueprint which will be followed as the child grows. The majority of children tend to resemble their parents. A notable exception occurs in the case in which the normal chromosomal structure is altered. This may be in misplacement, addition, or deletion of the genetic material.

Normal human cells, except for reproductive cells, contain thousands of gene pairs occupying specific positions on the 23 pairs of chromosomes. An offspring receives 23 chromosomes from each parent. Reproductive cells contain 23 chromosomes instead of the 46 found in all other cells. Twenty-two of the paired chromosomes (autosomes) have genes which are not sex-linked. The twenty-third chromosome pair (allosomes) contain the sex-linked genes, which determine the gender of the individual. Genetic transmission follows laws developed by Mendel. When each parent contributes one gene of a gene pair, there appears in the offspring one of four possible combinations: dominant gene from father and recessive gene from mother, dominant gene from father and dominant gene from mother, recessive gene from father and recessive gene from mother, and recessive gene from father and dominant gene from mother. Genes are transmitted from each parent, which accounts for the differences in traits exhibited by children. As an example, when dominant gene brown eyes are paired with recessive gene blue eyes, the individual will have brown eyes. Dominant genes include brown eyes, gray hair, and two conditions which result in mental retardation: tuberous sclerosis and neurofibromatosis. When two recessive genes or two dominant genes are paired, the manifested trait is affected by the factors of penetrance, expressivity, age of onset, and polygenic inheritance.

Penetrance refers to the proportion of people in a given population who exhibit a given genetic trait. Genes with low penetrance will not have their traits manifested by many people. Expressivity refers to the severity of the trait. Age of onset is important in genetic counseling. A parent may transmit a trait before the trait is manifested in the parent. Polygenic inheritance refers to the interaction among gene pairs. More than one gene pair may affect the appearance of a particular trait.

Recessive gene transmission occurs when two recessive genes are paired. Examples of this include blue eyes and

certain conditions which may cause mental retardation, including phenylketonuria, galactosemia, and Tay-Sachs disease.

Other characteristics are carried on the twenty-third chromosome pair. Gender of the offspring is determined by the father, who contributes the deciding chromosome. The female twenty-third chromosome is characterized by the gene pair XX and the male twenty-third chromosome takes the form of XY. If the father contributes an X chromosome, the pair is XX and the offspring is female. If the father contributes a Y chromosome, the pair is XY and the offspring is male. Sex-linked recessive traits are carried on the X chromosome and include color blindness, hemophilia, and Lesch-Nyhan syndrome.

Sometimes errors occur during reproduction. These are not hereditary, but occur at conception. Meiosis is the division of germ cells or gametes (ova and sperm cells) which requires two splits. Mitosis is the division of all other cells. Errors in mitosis or meiosis cause the gametes to end up with more than or less than their normal complement of chromosomes. Most chromosomal aberrations consist of one or more extra chromosomes in their cell nuclei. Other aberrations include the absence of chromosomal material and the reattachment of detached material to the wrong chromosomes. Aberrations may occur among the allosomes or autosomes. Most affected fetuses do not survive to term, but approximately one in 200 do and the prevalence of chromosomal aberrations is approximately one in 200 live births. About ten percent of nonfamilial retardation is caused by chromosomal abnormalities.

Autosomal Aberrations

The most widely known group of chromosomal aberrations is often subsumed under the label Down's syndrome. Autosomal chromosomes are composed of the twenty-two pairs which are not sex-linked. The twenty third pair (allosomes) are sex-linked and will be discussed below. Physical characteristics associated with Down's syndrome include short limbs, stubby fingers, an enlarged tongue which protrudes from the mouth, small teeth, low set ears, a highly arched palate, vision problems, husky voice, slanted eyes which are often accompanied by a fold of skin over the inner corner of the eyelid (epicanthic fold), flattened facial features and back of the head, and a tendency toward obesity. The Down's syndrome baby is "floppy" due to weak muscles which lack tone. About half of these infants have congenital heart defects. Children who have Down's syndrome are much more likely to suffer from acute leukemia. Except for those with dark brown eyes, the occurrence of Brushfield spots (light colored specks in the iris) is likely. Fingerprints, handprints, and footprints deviate from normal. Mental

retardation generally ranges from moderate to severe. The range of incidence has been reported as one in 600 to one in 900. Maternal age correlates with incidence as shown in the following table:

Maternal Age	Incidence
15-24	1 in 1500
25-34	1 in 1000
35-39	1 in 750
40-44	1 in 70
45 and over	1 in 20-38

Several forms of this syndrome can be manifested.

Nondisjunction

Nondisjunction occurs during meiosis or mitosis and results in an incomplete split of chromosomes. At conception, the resulting embryo has three chromosomes (trisomy) instead of the normal pair at one of the 23 pairs of chromosomes. The most widely known example of disjunction is called trisomy 21 or congenital acromicria. It is more correct to associate the term Down's syndrome only with this aberration, which was first described by Langdon Down in 1866. Confusion arises because diagnosis was based on physical features until recently. Diagnosis is now accomplished by cytogenetic procedures, which establish that in trisomy 21, disjunction occurs at the 21st pair, resulting in three #21 chromosomes in each cell instead of the normal two. In about three-fourths of the cases, the extra chromosome is contributed by the mother. Trisomy 21 children generally fall below average in height, weight, stature, and life expectancy. Because of better medical intervention and increased recreational opportunities which promote better fitness, life expectancy is steadily improving. Nondisjunctive trisomies have been reported at chromosome pairs 1 through 18, as well as #21. Trisomy 13 is called Patau's syndrome. It was described in 1960. Survival is rarely more than three years and all affected have been profoundly retarded. Trisomy 18 is called Edward's syndrome and was also described in 1960. Early death and mental retardation is also characteristic of trisomy 18.

Translocation

Translocation is an error which occurs when either a whole chromosome or a fragment of a chromosome attaches itself to another chromosome. This attachment may be to the chromosome's partner or to a chromosome of a different pair. It is possible that a trisomy 21 victim may have one of the

#21 chromosomes attach itself to a chromosome #15. This is called translocation trisomy and results in a child who demonstrates the symptoms of Down's syndrome. Because of the attachment to one of the #15 chromosomes, this child has the normal complement of 46 chromosomes.

Mosaicism

Mosaicism occurs after conception. During the second cell division, non-disjunction occurs, resulting in two normal cells, one cell with trisomy, and one with monosomy. The monosomic cell dies, leaving one trisomic cell and two normal cells. These cells continue dividing and the individual has some cells with 46 and some with 47 chromosomes. Manifestations depend on which body parts receive which cells in which proportions.

Deletion

In deletion, a portion of the original genetic material is absent from a chromosome. Cri du chat (cat cry) is the most known of the syndromes. It is caused by a partial deletion of chromosome #5. Infants manifest an eerie, mewing, cat-like cry and mental retardation is usually in the severe to profound range. Physical characteristics range from Down's-like to almost normal.

Allosomal Aberrations

When non-disjunction or variation occurs on the sex chromosomes (allosomes), intellectual and physical anomalies may be present in the survivors. Although rare, several types of sex-linked aberrations exist.

Turner's syndrome is also called gonadal aplasia. It was described in 1938 and affects about one in 5000 live births. Only females are affected because there is an absence of one of the sex chromosomes. It is possible to live without a Y chromosome, but not without an X. Since only one sex chromosome is present and it must be an X, only females survive (XO). Secondary sex characteristics fail to develop without estrogen therapy and mild mental retardation may or may not be present. There is a slight tendency toward visual-perceptual deficits and nerve deafness. Turner's syndrome can also include mosaic individuals who have a proportion of normal cells.

Klinefelter's syndrome was described in 1942. Males who are affected generally have one extra X chromosome (XXY), but it is possible to have up to three extra X chromosomes. It is even possible to have one extra Y and one extra X chromosome (XXYY). Poly X men develop female breasts, do not produce sperm, and have small testes. The relationship to mental retardation is not yet completely clear, but it

appears that the frequency and degree of mental retardation increases as the number of extra chromosomes rises. Poly X females may have up to three extra X chromosomes. One extra X chromosome is not associated with mental retardation, but if the female has two or three extra X chromosomes, mental retardation is consistently present.

The fragile X chromosome is also called the satellited X or marker X. It is a variation in which a small appendage appears on the end of the long arm of the X chromosome. Females may act as carriers. Fragile X males are mentally retarded and have enlarged testicles (macroorchidism). As many as one in 850 live births could be affected.

VII Gestational Disorders

Discoveries about the etiology of mental retardation are decreasing the need for generic categories such as gestational disorders and unknown prenatal influences. As causal factors are investigated and described, syndromes can be placed in appropriate categories such as infection and intoxicants, trauma or physical agent, metabolism or nutrition, and chromosomal abnormality. Since gestational disorders cover the same period of time (i.e., pregnancy) as unknown prenatal influences, it probably would be wise to collapse them into a single category.

Included in gestational disorders are the associated factors of prematurity and low birth weight. The relationships among prematurity, low birth weight, diet, environmental factors, socio-economic-status (SES), and infection are complicated. It is difficult to study cause and effect relationships because of confounding social variables. Ethically, it is not possible to isolate and study variables such as malnutrition. Research in this area often is only possible on a post hoc (after the fact) basis.

Prematurity is most closely associated with mental retardation and other high risk categories. Poor diet, including poor maternal nutritional history, accounts for a higher rate of prematurity among the disadvantaged than any other cause. Another factor in prematurity is excessive maternal cigarette smoking. Premature babies are more likely to experience neonatal lung collapse, hyaline membrane disease, and hyperbilirubinemia.

About one in seven successful pregnancies result in low birth weight babies (less than 2500 grams or 5 and 1/2 pounds), with up to half caused by fetal malnutrition. In addition to obvious causes, fetal malnutrition may result from maternal stress, which causes decreased uterine blood flow. Fetal malnutrition is synonymous with small for gestational age, small-for-date, and growth retardation in utero. A reduced number of brain cells, slower rate of CNS cell division, and reduced amounts of DNA in the cells are associated with low birth weight.

VIII Retardation Following Psychiatric Disorder

When children with normal intelligence experience conflicts with reality, they may earn IQ scores in the range of mental retardation. Coupled with the bizarre behavior they exhibit, it is possible to argue that they meet the criteria for mental retardation as defined by the American Association of Mental Deficiency and federal law.

Because they appear to function as mentally retarded children, it is reasonable to assume that they should be placed in classes for the mentally retarded. In this way, they would be taught at their functional level with other students who perform in similar manner.

Others would argue that their IQ scores are invalidated by their difficulties in affect and that their maladaptive behavior is based on an inability to resolve the difference between self-perception and reality, rather than an inability to comprehend in the cognitive realm. Using this qualitative difference as a basis for placement in classes for the mentally retarded is thought by many to not be in accord with the spirit behind the term impairment in adaptive behavior.

This issue will continue to be debated and may not be resolved with current knowledge. It is possible that the need for this category will diminish to the point that it will be eliminated.

IX Environmental Influence

Mental retardation is commonly divided into two broad categories--organic and familial. Organic retardation occurs when the CNS is damaged and includes all categories in this classification system except the last two, Retardation Following Psychiatric Disorder and Environmental Influence.

Environmental influence is synonymous with familial retardation or cultural familial retardation. Other terms which have also been used to describe this condition include simple amentia, subcultural mental deficiency, garden-variety mental deficiency, and endogenous mental deficiency.

Over eighty percent of students classified as mentally retarded fall into the cultural familial classification. There is no biologically known cause for the retardation, yet these learners fail to meet normal learning expectations.

One explanation is that intelligence is distributed in the population in a fashion similar to other empirically studied phenomena. The distribution of intelligence takes the form of a bell shaped curve in which there always will be people at the lower end. Mental retardation, then, is a statistical phenomena, i.e., the people on the bottom end of the IQ distribution. When those retardates classified as organic are eliminated from the distribution, the bulge at the lower end of the curve disappears, resulting in an almost perfectly shaped bell distribution.

Cultural deprivation, low maternal educational level, sensory deprivation, psychological maltreatment, environmental deprivation, and low socio-economic-status have been associated with cultural familial retardation. It is thought that marginal children are pushed into mental retardation when they are raised in a deprived environment. Deprivation may occur when family affect is low, when there is a pervasive feeling of despair, when the child does not have the benefit of examples of rich language structure and elaborate language patterns, when remedial or preventive services are inadequate or inaccessible, when parents are not sufficiently involved, and when children are not adequately taught and reinforced for expected behaviors.

The deprivation hypothesis, however, may be a simplistic explanation. Cultural familiar retardation is not confined to children normally thought of as deprived. The issue is complex because of the multiplicity of psychological variables involved and the relative lack of sophistication with regard to the ability to diagnose subtle organic problems.

Functioning Level

Besides mental retardation, cerebral palsy, epilepsy, and autism may cause functional limitations to the point that life chances will be diminished. The federal government uses the term developmental disabilities to characterize those with severe, chronic disability attributable to mental or physical impairment or combination manifested before age twenty-two, likely to continue indefinitely, and result in substantial functional limitations in three or more areas of major life activity. Examples of major life activity include self-care, receptive and expressive language, learning, mobility, self-direction, capacity for independent living, and economic self-sufficiency.

CHARACTERISTICS OF THE MENTALLY RETARDED

Four levels of mental retardation exist. Whether these levels indicate qualitative or quantitative differences in performance is debatable. The mildly mentally retarded are closest to regular education. Moderately mentally retarded children can learn to read at low levels. Severely and profoundly retarded learn rudimentary skills at best. Early intervention and research efforts continue to improve the educational prognosis for retarded learners to the point that expectations continually have to be revised upward.

Mild Mental Retardation

Mild mental retardation is also known as educable mental retardation (EMR). In past years an affected person might

have been called a moron, a high grade defective, marginally independent, or semi-independent. Roughly ninety percent of the retarded fall into the category of mild mental retardation and about eighty-five percent of the mildly retarded are classified as cultural familial.

The IQ level of this group is defined as -2 to -3 standard deviations below the mean on a standardized intelligence test. This ranges from a high of 68-70 to a low of 52-55. IQ tests were designed to predict a child's ability to function in school. As a rule of thumb, it is said that the EMR student will have a functioning level or rate of progress about one-half to two-thirds that of normal. This rule is based on the assumption that since the IQ's of the mildly retarded are approximately one-half to two-thirds those of the normal individual, their level of functioning should likewise be one-half to two-thirds that of the normal individual. Caution should be used in applying this rule of thumb. It should be emphasized that the IQ scores of the mildly retarded have a range of approximately twenty points and that there is a comparable range of scores for those individuals considered normal. Any position taken about a student based on IQ should be tentative and subject to change.

Members of this group are usually not identified until they have academic difficulty in school. Their appearance and behavior appear to be in the normal range, except that they are slightly behind in academics when compared to their normal peers. As time passes, they fall further and further behind.

The educational characteristics of the mentally retarded have been discussed at length by a number of educators. Some would characterize the development and learning styles of the retarded as being the same as those of the nonretarded. The only differentiation is that the retarded tend to function in a similar manner to chronologically younger nonretarded children. Adoption of this position would lead to the conclusion that no special insights and conceptual framework are necessary for teaching and handling this population. In practice, the educator would treat the twelve year old mildly retarded student as if the student were a normal eight year old. The danger inherent in this position is that generalizations from academic behavior to nonacademic behavior may be made.

Another position is that mental functioning is just one variable making up a person and that emotional status, physical status, and other developmental factors must be considered in the educational placement of the mildly retarded student. The practitioner would consider factors other than IQ (and mental age) when designing an instructional program for the student.

Goals for the mildly retarded are the same as those for the nonretarded. The areas of import are: (1) basic

academic skills, (2) personal and social competence, and (3) occupational skills. To meet those goals, programming for the EMR student has traditionally taken one of three forms:

1. The EMR student follows the regular curriculum, taking longer to finish. This is based on a quantitative explanation of their disability. They are slower and, therefore, it takes longer to attain acceptable levels of competence. At the age when their nonretarded peers graduate from high school, the EMR student is expected to have met second to eighth grade goals and objectives. Proponents of this approach often argue that the EMR student should remain in school until the age of twenty-one or beyond. It is thought that this extra time is needed to catch the EMR student up to his nonretarded peers.

2. The EMR student follows a less rigorous form of the regular curriculum, finishing with his nonretarded peers. This is a rational response to the belief that the EMR student learns in a fashion qualitatively different from his nonretarded peers. Often called the "watered-down curriculum," it allows the EMR student the advantage of staying closer to his nonretarded peers throughout his school career.

3. The EMR student follows a curriculum in which the emphasis given to various goals is changed. This recognizes that the EMR student has needs which can better be met with curriculum modifications. One model emphasizes personal, social, and occupational goals at the expense of academic goals.

Conventional thought is that the mentally retarded can more easily acquire motor and social skills. Academic skills, therefore, can be better acquired through training in industrial arts, homemaking skills, and arts and crafts. The idea of a functional curriculum built around life chances is very appealing. As an example, units in child care would be offered at various levels. In the preschool years, the unit might center around the care of puppies. At the primary level, reading is taught as a functional skill which will help the students to learn to feed puppies correctly. Academics are introduced as the students become ready. At the secondary level, a unit might center around nutrition to be a good parent.

No matter what the emphasis, the expectation is that the EMR student will leave school with the skills necessary for social and economic self-sufficiency. These expectations are defined in the following chart:

Sociability--able to function in society

Citizenship--able to obey laws and conform to legal norms

Parent and Adult Responsibilities--well adjusted

Economic Self-Sufficiency--supports self and family

Communication--makes needs and wants known to correct source, which usually requires fourth grade literacy

Leisure Time Use--avoids difficulty
--uses leisure time constructively.

Some would feel that schools have done their job successfully when the EMR student becomes an unremarkable adult.

The mildly retarded child enters school with characteristics which must be considered in program design. Modifications can often be carried out in the regular classroom, which will meet the needs of this child.

In the area of language, the performance of the mentally retarded is often below that of their mental age. Speech anomalies occur frequently, with poor articulation, voice disorders, and stuttering posing the most problems. With respect to speech problems, EMR children are not substantially different from the nonretarded population. The language of the mentally retarded may be characterized as primitive and immature.

In learning ability, the EMR student is faced with deficits in attention, memory, transfer of training (generalization), learning set, and problem solving. The EMR student has difficulty attending to a variety of stimuli. He has a short attention span compared to his normal peers and takes longer to "catch on" because he is easily distracted.

In the area of memory, long term memory may not be any different from normal, but short term memory is poor. Mildly retarded children tend to forget more quickly, especially without practice. This may be attributed to the fact that the EMR child often fails to employ appropriate learning strategies, particularly in the area of mediation. One example of a mediation strategy is rehearsal. Instead of naturally learning how to practice to themselves, EMR children must be shown and continually reminded until the process becomes internalized. Another learning strategy which is difficult for the EMR child to master is organization. Clustering is an organizational strategy in which the child must rehearse serially presented items in groups. EMR children must learn to classify items into groups, chunk items in each group, learn the chunks, and string the chunks together into a whole.

Another characteristic of note is that mentally retarded students tend to have problems in transferring what is learned in one situation to another. Practice in various situations can help to improve transfer of training ability.

Much is made of the inability of EMR students to learn in an abstract fashion. It is better to teach them in a concrete fashion through the use of manipulatives. Experienced practitioners also make sure that the increments

between objectives are kept small enough so that the objectives may be more easily mastered.

Another problem involves incidental learning. EMR students have difficulty paying attention to materials, concepts, and processes which are not directly presented. A highly structured approach insures that attention will be paid to that which is important to the learner's success.

Mildly retarded students also seem to be deficient in their ability to adopt a learning set, which is important in solving problems. Problem solving and other skills must be carefully and painstakingly taught. A great deal of emphasis must be placed on insuring that the student has mastered each skill before moving on to the next objective in the curriculum.

In academic achievement, EMR students tend to have difficulty in all areas, with reading presenting the major problems. Although their academic progress is slow, most can master the basics of the academic curricula. The progress of the EMR student in nonacademic areas such as athletics, mechanics, or the arts can be normal. Care should be taken not to equate potential for academic success with potential for nonacademic success.

In social adjustment, EMR children demonstrate an inability to avoid being manipulated by others. This is described in the literature as being other-directed, or outer-directed, or having an external locus of control. Poor motivation, negative self-concepts, and passivity have all been associated with this group. The mildly retarded are not expected to overcome self-centeredness, develop social insights and the ability to make moral judgments, refine social problem solving skills, and develop social relations abilities at the same rate as their nonretarded peers.

Research has shown that EMR children who are left in the regular classes tend to become academically more proficient than those who remain in self contained classes. Conversely, those who remain in self contained classes tend to develop better social skills and adjust better to the vocational demands of adult life. If done correctly, the mix of special and regular educational programming which most EMR students should receive offers them the best chance for successfully meeting the demands of adult life.

Goals

At the preschool level, the difference is that EMR children take two to three years to acquire skills usually acquired in one year by normally functioning children. Hallahan and Kauffman (1982) stated that preschool children need to learn to:

1. Sit still and attend to the teacher.

2. Discriminate auditory and visual stimuli.

3. Follow directions.

4. Develop language.

5. Develop gross and fine motor skills, including holding a pencil and cutting with scissors.

6. Develop self-help skills, including tying shoes, buttoning and unbuttoning, zipping and unzipping, and toileting.

7. Develop the ability to interact with peers in group situations.

At the primary level, there is a shift toward more academic skills while preschool skills are enhanced

1. Emphasize gross motor skills such as crawling, walking, running, jumping, skipping, throwing, and balancing.

2. Emphasize readiness or fine motor skills such as cutting, coloring, folding, threading, bead stringing, and drawing.

3. Emphasize language development.

4. Emphasize concept formation

5. Begin reading. It is important to emphasize comprehension.

6. Begin arithmetic. EMR children have attention, abstract thinking, transfer, comprehension, observation, initiative, and pacing problems which hamper them in this area.

7. Begin handwriting. The goal is legibility in personal expression.

8. Begin making decisions, i.e., selecting among stated options.

At the intermediate level, more stress should be placed on academics.

At the secondary level, a shift towards applied academics is warranted.

1. Read newspapers, street signs, labels on products

2. Make change to $1.00, $10.00, $100.00, etc.

3. Fill out job application forms.

The transition from school to work is difficult for most students. At the secondary level, an individualized plan must be developed for each student to address the issues of transition. Vocational goals for mildly retarded students range from competitive employment, employment on work crews or enclaves, to sheltered work arrangements. Training to meet those goals can occur in: vocational-technical settings, vocational rehabilitation centers, vocational education courses, industrial and business settings, community service and nonprofit organization work settings, school or campus settings, sheltered workshop settings, and work activity settings. The emphasis for the EMR student should be on preparation for competitive employment.

Managing the EMR Child in School

1. Sequence learning tasks into a series of steps and teach one step at a time.

2. Drill and repetition are often necessary.

3. Encourage verbal mediation, i.e., get them to rehearse.

4. Increase motivation--combine novelty with structure and familiarity.

5. Develop school-related behaviors such as task completion and independent work habits. Teach them to learn.

6. Continuous assessment will help planning.

7. Continuous and immediate feedback is important.

Moderate Mental Retardation

About six percent of the retarded population are moderately mentally retarded. Students at this level are often called trainable mentally retarded (TMR). A much greater proportion of this subpopulation is traced to an organic etiology, e.g., Down's syndrome. This group functions at about one-third to one-half the normal rate. There is less emphasis on academic skills and more on functional skills. Teaching is concrete, direct, and repetitive.

Goals

1. Self-help: toileting, dressing, feeding, and grooming.

2. Communication: speech, language, listening, and nonverbal skills.

3. Personal-social: interactive skills, personal maintenance, and self-judgment programming.

4. Perceptual-motor: visual, auditory, haptic discrimination; eye-hand coordination; balance; fine and gross motor skills as ends in themselves.

5. Functional Academic skills: to function in society; reading--names, directions, and labels; writing--pertinent words and names; arithmetic--rote counting, number recognition, time, simple computational skills, and simple money exchange.

6. Vocational skills: semi-independent skills

Vocational goals for moderately retarded students range from competitive employment, employment on work crews or enclaves, to sheltered work arrangements. Training to meet those goals can occur in: vocational-technical settings, vocational rehabilitation centers, industrial and business settings, community service and nonprofit organization work settings, school or campus settings, sheltered workshop settings, and work activity settings. The emphasis for the TMR student should be on preparation for work crews or enclaves.

Managing the TMR Child

Hendrick (1980) provides a number of teaching tips for younger children. These include: treating the TMR child as much as possible as nonretarded, awareness of developmental expectations, frequent repetition, teaching behavior which is appropriate at later developmental periods, allowing extra time, teaching persistence, encouraging independence, teaching one skill at a time, using a hands-on approach, encouraging speech production, watching the affective climate, displaying positive affect, and being ready to abandon a task which is too difficult until a later time.

Severe/Profound Mental Retardation

About three percent of the retarded are severely retarded and about one percent are profoundly retarded. There is a wide range of behavior in each of these categories. Training emphasizes basic survival and self-help skills, along with eliminating undesirable behaviors such as rocking, scratching, eye rubbing, etc.

Goals

1. Sensori-motor stimulation

   A. Preschool
      Stimulate sight, hearing, touch, smell, and muscular response
      Enrich the environment and encourage exploration of interesting and attractive surroundings

   B. School-aged
      Identify shapes, colors, sizes, locations, and distances
      Identify sound patterns, locations, tonal qualities, and rhythms
      Identify textures, weights, shapes, sizes, and temperatures
      Identify familiar, aversive, and pleasant odors

   C. Adults
      Sorting, transferring, inserting, pulling, and folding
      Responding to music activities, signals, and warnings
      Making personal choices and selections
      Discriminating sizes, weights, colors, distances, locations, odors, temperatures, etc.

2. Physical
   A. Pre-school; stress development
      Body positioning
      Passive exercising
      Rolling, creeping, and crawling
      Balancing head and trunk
      Using hands purposefully
      Practice standing
      Training for mobility
   B. School age; stress mobility and coordination
      Practice ambulation
      Overcoming obstacles, walking on ramps and stairs, running, skipping, jumping, balancing, and climbing
      Using playground equipment
      Participating in track and field events
   C. As adults; stress dexterity and recreation
      Riding vehicles, participating in gymnastic-like activities and track and field events
      Marking with pencil, cutting with scissors, stringing beads, pasting, and assembling
      Swimming and water play
      Using community parks, playgrounds, and other recreational resources

3. Self-care
   A. Preschool
      Taking nourishment from bottle and spoon, drinking from cup and finger feeding
      Passive dressing, accommodating body to dressing, partially removing clothing
      Passive bathing, handling soap and washcloth, participating in drying
      Passive placement on toilet, toilet regulating
   B. School-aged
      Self-feeding with spoon and cup, eating varied diet, behaving appropriately while dining
      Removing garments, dressing and undressing with supervision, buttoning, zipping, and snapping
      Drying hands and face, partially bathing
      Toilet scheduling, indicating need to eliminate, using toilet with supervision
   C. Adults
      Eating varied diet in family dining situation, using eating utensils, selecting foods
      Dressing with partial assistance or supervision
      Bathing with partial assistance or supervision
      Using toilet independently with occasional supervision

4. Language
   A. Preschool; stress stimulation
      Increasing attention to sounds
      Encouraging vocalization
      Responding to verbal and nonverbal requests
      Identifying objects
   B. School age; stress development
      Recognizing name, names of familial objects, and body parts
      Responding to simple commands
      Initiating speech and gestures
      Using gestures, words, or phrases
   C. Adult; stress development
      Listening to speaker
      Using gestures, words, phrases
      Following uncomplicated directions

5. Social/vocational
   A. Preschool; stress interpersonal response
      Recognizing familiar persons
      Requesting attention from others
      Occupying self for brief periods
      Manipulating toys or other objects
   B. School-aged; stress social behavior
      Requesting personal attention
      Playing individually alongside other children
      Using basic self-protective skills

Playing cooperatively with others

C. Adult; stress self-direction and work
Using protective skills
Shaving, taking turns, waiting for instructions
Traveling with supervision
Completing assigned tasks
Participating in work activity center programs.

Behavior modification techniques may be used effectively with this population. Chaining, reverse chaining, shaping, fading, and imitation have been successfully used.

Gaylord-Ross and Holvoet (1985) have proposed a functional curriculum model which incorporates eight components:

1. Select functional materials and activities.

2. Teach in functional settings using natural cues whenever possible.

3. Use varied materials and allow students to choose materials whenever possible.

4. Incorporate communication programs into natural, functional routines.

5. Incorporate motor programs into natural, functional routines.

6. Sequence skills in a logical and normative manner.

7. Incorporate deceleration programs into skill sequences.

8. Teach in groups as well as one-to-one settings. (p. 93)

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American Journal of Mental Deficiency.

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